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Review

Epistaxis During Antiplatelet and Anticoagulant Therapy in Patients with Cardiovascular Disease: Clinical Dilemmas, Guideline Comparison, and a Personalized Management Algorithm

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Abstract

Background and Objective. Epistaxis is a frequent bleeding manifestation in patients receiving antithrombotic therapy, and its management falls between otolaryngology and cardiology guidance. We aimed to translate the evidence into an operational framework for joint decisions. Methods. PubMed/MEDLINE, Scopus, Embase, and the Cochrane Library were searched (January 2015 through August 2026), current ESC, ACC/AHA, EHRA, AAO-HNS, and SFORL guidance were analyzed, and national clinical protocols of different countries were screened; 67 sources were included. Results. Direct oral anticoagulants were consistently associated with fewer clinically relevant epistaxis events than warfarin, and minor epistaxis led to permanent anticoagulant discontinuation more often than minor bleeding at other sites. No guideline defines epistaxis severity, and guidance diverges on protected dual antiplatelet therapy, bleeding-risk assessment, and the sequence of local hemostasis and drug interruption. We propose a three-tier severity classification (minor, clinically relevant non-major, major) informed by ISTH and BARC definitions, a pragmatic thrombotic risk stratification, and an algorithm with agent-specific decisions. Conclusions. In minor and most clinically relevant non-major episodes, antithrombotic therapy can usually be continued while local hemostasis is achieved; interruption, de-escalation, or reversal should be individualized and reserved for uncontrolled or major bleeding. Effective local hemostasis can help avoid unnecessary interruption of antithrombotic therapy, and otolaryngologists should be part of multidisciplinary teams managing such bleeding.

Keywords: epistaxis; anticoagulants; antiplatelet agents; atrial fibrillation; bleeding classification; tranexamic acid; clinical guidelines

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1. Introduction

Epistaxis is among the most frequently encountered mucosal bleeding manifestations in patients receiving antithrombotic therapy, although its relative frequency varies with the agent, the population, and the bleeding definition used [1,2]. Population aging, the rising prevalence of atrial fibrillation [3] and coronary artery disease, and the widespread adoption of contemporary dual antiplatelet therapy regimens [4,5] and direct oral anticoagulants have increased the frequency with which otolaryngologists encounter epistaxis in patients with cardiovascular disease. An association between epistaxis and atherosclerotic vascular disease has also been reported in this population [6].

In phase 3 trials of rivaroxaban, epistaxis accounted for 8–16% of all treatment-emergent adverse events, a denominator that is broader than bleeding events alone [7]. Among 600 presentations with active epistaxis, 66.8% of patients were receiving antithrombotic therapy (oral anticoagulants, antiplatelet agents, or their combinations) [8], whereas in a teaching-hospital series of 721 patients with epistaxis, 78% were taking either an antiplatelet or an anticoagulant agent [9].

This creates a challenging clinical scenario for practicing physicians: ongoing bleeding requires prompt intervention, whereas unjustified interruption of antithrombotic therapy increases the risk of stroke, stent thrombosis, or systemic embolism, events that may be more dangerous than the hemorrhage itself [10]. A further difficulty is that management is governed by guidance from two specialties: otolaryngology guidelines provide detailed recommendations on local hemostasis but give limited attention to the subsequent management of antithrombotic therapy, whereas cardiology guidelines comprehensively address antithrombotic treatment but do not consider epistaxis as a distinct clinical scenario.

The aim of this narrative review was to systematize contemporary evidence on the incidence, severity, and outcomes of epistaxis during antiplatelet and anticoagulant therapy, compare current guideline recommendations, define operational severity criteria for epistaxis in this population, and propose a practical management algorithm.

2. Materials and Methods

A literature search was conducted in PubMed/MEDLINE, Scopus, Embase, and the Cochrane Library for the period from January 2015 through August 2026; the last search update was run at the end of August 2026, before the manuscript was submitted in September 2026. The keywords "epistaxis" and "nosebleed" were combined in various ways with "anticoagulant," "antiplatelet," "direct oral anticoagulant," "vitamin K antagonist," "atrial fibrillation," "dual antiplatelet therapy," and "tranexamic acid." Current clinical guidelines and consensus documents were also identified on the websites of the issuing societies (ESC, EHRA, ACC/AHA, AAO-HNS, and SFORL), national clinical protocols of different countries were sought on the websites of national health authorities, and the reference lists of included publications were screened. Publications in English were considered. The database search was limited to January 2015–August 2026; guidelines, consensus documents, and sources identified through citation searching (including earlier bleeding definitions, risk scores, and landmark studies) were not restricted by publication year.

Inclusion criteria were studies addressing epistaxis in adults receiving antiplatelet or anticoagulant therapy; clinical guidelines and consensus documents on the management of bleeding during antithrombotic therapy; and studies of local hemostatic methods applicable to this patient population. For each guidance document, the most recent version in force at the time of the last search was used. Recommendations were extracted by a cardiologist and an otolaryngologist into 12 predefined clinical domains and discrepancies were resolved by consensus within the author team. Exclusion criteria were hereditary hemorrhagic telangiectasia and other inherited vascular dysplasias; tumors of the nasal cavity, paranasal

sinuses, or skull base; perioperative and postprocedural bleeding; epistaxis reported as an adverse event of drugs from other pharmacological classes; congenital coagulopathies and platelet disorders; and pediatric populations unrelated to antithrombotic therapy.

The literature search identified 318 records. After removing 11 duplicates, 307 records were screened, of which 233 were excluded. Seventy-four full-text reports were assessed for eligibility, and seven were subsequently excluded. Consequently, 67 studies were included in the final narrative review (Figure 1). The 318 records comprised 307 records from databases and 11 records identified through other methods (guidelines, national clinical protocols, and citation searching); the arithmetic of each step is 318 − 11 = 307, 307 − 233 = 74, and 74 − 7 = 67. All 67 included sources are cited in the reference list. The flow diagram is provided to ensure transparent reporting of the search methodology in accordance with PRISMA 2020 [11] and is not intended to imply formal compliance with the requirements for systematic reviews.

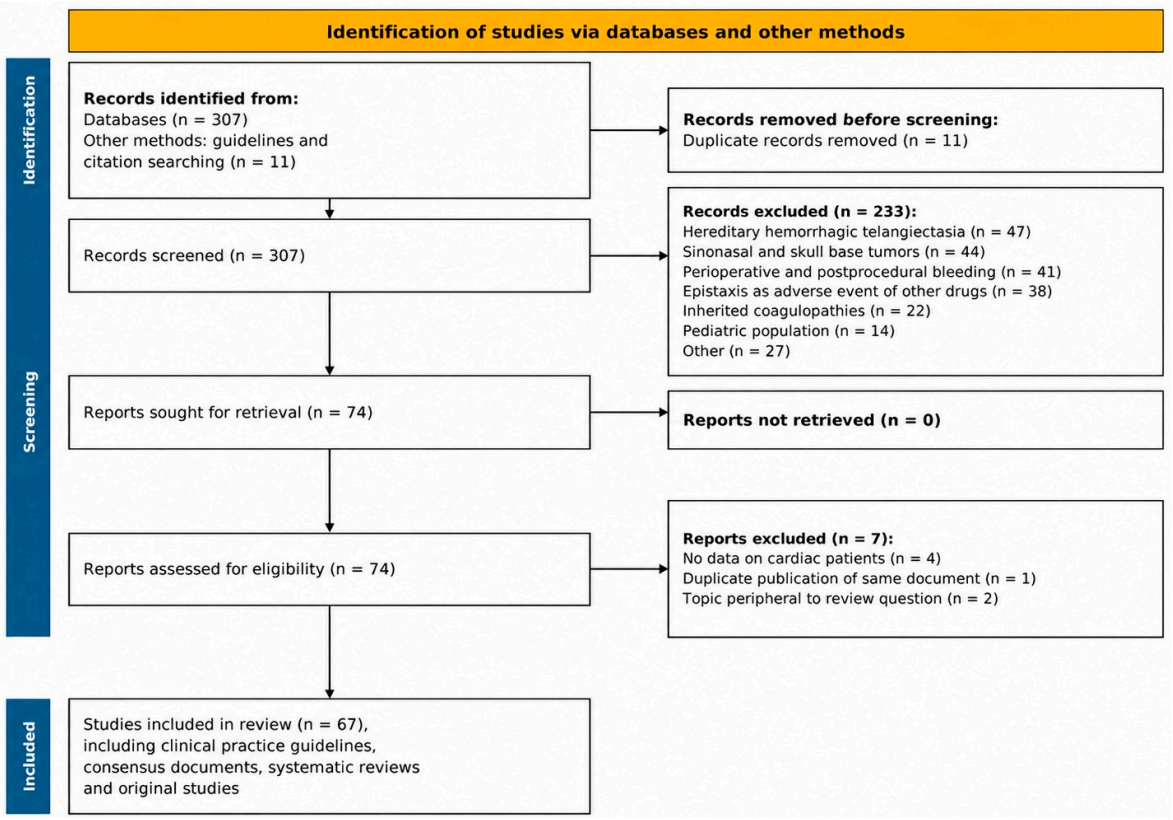


Figure 1. Flow diagram of literature identification and selection, adapted from the PRISMA 2020 template [11]. Database search period: January 2015-August 2026; sources identified through other methods (guidelines, national clinical protocols, and citation searching) were not restricted by publication year. Of 318 records identified, 67 sources were included in the narrative review; all are cited in the reference list.

This work is a narrative review: the protocol was not registered, and no formal risk-of-bias assessment or meta-analysis was performed. Reporting was structured in accordance with the SANRA criteria [12].

Definition and Classification of Epistaxis Used in This Review

In this review, epistaxis is defined as bleeding from the vessels of the nasal cavity, paranasal sinuses, or nasopharynx. It is classified along two independent axes: the location of the bleeding source and the severity of bleeding.

By location, anterior and posterior epistaxis are distinguished [13]. The distinction matters in practice: in a prospective study of 50 consecutive adults with idiopathic posterior epistaxis, the bleeding source was identified by endoscopy without general anesthesia in 94% of cases, and 70% of the identified sources were located on the nasal septum [14].

No generally accepted definition of epistaxis severity exists. The AAO-HNS guideline states that there is no standard definition of severe epistaxis [13], and the multidisciplinary consensus of the British Rhinological Society covers epistaxis of any severity without stratifying it [15]. Because the target population of this review consists of patients receiving antiplatelet or anticoagulant therapy, severity was operationalized with reference to validated bleeding definitions used in cardiovascular and anticoagulation research (Table 1) [16–20]. Table 1 was developed jointly by the cardiology and otorhinolaryngology members of the author team. Throughout the manuscript, the terms minor, clinically relevant non-major (CRNM), and major epistaxis are used in this sense.

Table 1. Operational definition of epistaxis severity applied in this review.

Severity	Operational Criteria	Closest Validated Definition
Minor	Stops spontaneously or with external compression of the nasal alae; no medical intervention required; no change in hemodynamic or laboratory parameters.	Below the ISTH threshold for clinically relevant non-major bleeding [17]
Clinically relevant non-major (CRNM)	Requires medical intervention (cautery of the source or anterior or posterior nasal packing) or an unscheduled medical contact, without meeting the criteria for major bleeding. Supporting markers: cumulative duration of more than 30 min within 24 h; three or more recent episodes.	ISTH clinically relevant non-major bleeding [17]; duration and recurrence markers as cited by AAO-HNS [13]
Major	Any of the following: hemoglobin decrease of 20 g/L or more; transfusion of 2 or more units of red blood cells; hemodynamic compromise requiring intervention, including intravenous vasoactive agents; need for surgical or endovascular hemostasis (arterial ligation or embolization); airway compromise; fatal bleeding.	ISTH major bleeding [16]; BARC type 3 [18]; ACC 2020 expert consensus [20]; hemodynamic criterion as in GUSTO [19]

AAO-HNS, American Academy of Otolaryngology-Head and Neck Surgery; BARC, Bleeding Academic Research Consortium; GUSTO, Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries; ISTH, International Society on Thrombosis and Haemostasis. The categories are pragmatic operational definitions for epistaxis and are not validated equivalents of the cited bleeding definitions.

Estimated blood loss was not used as a criterion, because in epistaxis it cannot be measured directly: part of the blood is swallowed, and estimates remain approximate.

The decision to discontinue, reduce, or reverse antithrombotic therapy is recorded separately, because it is not captured by any of these scales and, in this population, it is this decision that carries the thrombotic risk. In line with expert consensus, it should follow a comparison of thrombotic and bleeding risk rather than the characteristics of the bleeding alone [20,21].

3. Incidence, Severity, and Outcomes of Epistaxis During Antithrombotic Therapy

3.1. Incidence Across Treatment Regimens

Most data on the incidence of epistaxis have accumulated over the past five years and are summarized in Table 2. The most robust quantitative estimates come from two independent cohorts. In a nationwide Icelandic propensity score-weighted study, warfarin was associated with a higher rate of clinically relevant epistaxis than direct oral anticoagulants, with a more than fourfold difference compared with apixaban (hazard ratio, 4.22; 95% CI, 2.08–8.59) [1]. In the MAQI^2 registry, a similar pattern was observed: 2.06 versus 0.75

emergency department visits for epistaxis per 100 patient-years [7]. These findings are shown in Figure 2 and are consistent with a meta-analysis [22] and earlier observational studies [23,24].

Table 2. Incidence, severity, and outcomes of epistaxis during antiplatelet and anticoagulant therapy.

Study, Design	Population and Period	Main Findings
Ingason A.B. et al., 2022 Nationwide cohort study [1]	Iceland, 2014–2019; 7081 patients	Rate of clinically relevant epistaxis per 100 patient-years: warfarin, 2.2; rivaroxaban, 1.0; apixaban, 0.6. Hazard ratio for warfarin vs. apixaban, 4.22 (95% CI, 2.08–8.59). INR exceeded 3.0 in one-third of patients receiving warfarin.
Burke K.S. et al., 2024 MAQI^2 registry analysis [7]	United States; atrial fibrillation and venous thromboembolism; 10,557 patients on warfarin and 3889 on DOACs	Emergency department visits for epistaxis: warfarin, 2.06 vs. DOACs, 0.75 per 100 patient-years ($p < 0.0001$). Hospital admission for epistaxis, 0.50 vs. 0.13 per 100 patient-years.
Semco R.S. et al., 2025 Post hoc analysis of ENGAGE AF-TIMI 48 [2]	Atrial fibrillation; edoxaban vs. warfarin; 21,105 patients, 1008 with epistaxis	Major bleeding among epistaxis events, 3.2% vs. 20.7% among other bleeding events. Permanent discontinuation after minor epistaxis, 33.3% vs. 23.9% ($p = 0.001$). Lower-dose edoxaban reduced the risk of epistaxis (hazard ratio, 0.73; 95% CI, 0.62–0.86).
Yaniv D. et al., 2021 Retrospective analysis [25]	470 emergency department patients with epistaxis, 2012–2018	Risk of severe bleeding: odds ratio, 1.8 ($p < 0.05$); hospitalization: odds ratio, 2.17 ($p < 0.05$). Recurrence rates did not differ and were 15%.
Zloczower E. et al., 2024 Retrospective cohort study [26]	786 patients with acute epistaxis, 2014–2022	Minor interventions were more frequent in the antiplatelet, DAPT, and vitamin K antagonist groups than in controls (74.4%, 79.6%, and 77.3% vs. 63.1%; $p < 0.05$); major interventions were more frequent with DAPT (5.6% vs 1.3%; $p = 0.053$). DOAC recipients did not differ from controls and required no major interventions. After multivariable adjustment, age was the only independent predictor of the need for a minor intervention.
Buchberger A.M.S. et al., 2018 Retrospective analysis [8]	600 emergency presentations with active epistaxis (521 patients), Germany, 2014	Any antithrombotic therapy, 66.8% (oral anticoagulants, 31.7%; antiplatelet agents, 16.7%; combinations, 18.5%). Oral anticoagulation was associated with recurrence ($p = 0.014$) rather than with a severe course.
Carey B., Sheahan P., 2018 Review of 721 cases [9]	721 patients presenting with epistaxis to a teaching hospital over 1 year	An antiplatelet or anticoagulant agent was used by 78%; arterial hypertension was present in 69.5%; treatment was successful in 89%.
Andersen B. et al., 2025 TriNetX database analysis [27]	United States, 2014–2024; adults with epistaxis, 18,550 without and 9530 with anticoagulation	After demographic matching (5480 in each cohort), anticoagulated patients had a higher risk of subsequent inpatient care (risk ratio, 5.99; 95% CI, 4.31–8.33) and emergency care (2.70; 2.39–3.03). These outcomes reflect all-cause healthcare utilization after the index episode, not epistaxis recurrence.

The first three studies report the incidence of epistaxis in populations receiving anticoagulants; the remaining studies describe patients presenting with epistaxis, among whom antithrombotic use and outcomes were analyzed. DAPT, dual antiplatelet therapy; DOAC, direct oral anticoagulant; INR, international normalized ratio. The principal conditions and contributing factors associated with epistaxis are summarized in Table S1, while an integrated clinical pathway for the management of epistaxis during antithrombotic therapy is presented in Figure S1 (Supplementary Materials).

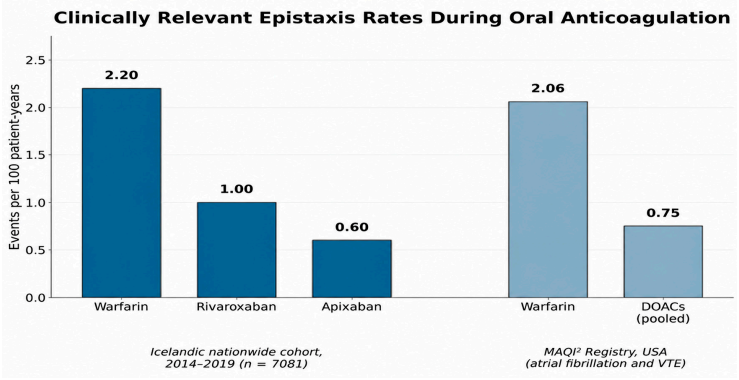


Figure 2. Rates of clinically relevant epistaxis during oral anticoagulation in two independent cohorts.

3.2. Severity and Impact on Treatment Persistence

The severity and outcomes of epistaxis differ from those of bleeding at other sites. In a post hoc analysis of the randomized ENGAGE AF-TIMI 48 trial, 4.8% of patients experienced epistaxis, accounting for nearly one in five extracranial bleeding events. Major bleeding represented 3.2% of epistaxis events versus 20.7% of bleeding events at other sites. The overall rate of permanent anticoagulant discontinuation did not differ; however, in the subgroup with minor bleeding, discontinuation after epistaxis was significantly more frequent (33.3% vs. 23.9%; $p = 0.001$) [2]. In other words, although epistaxis was on average less severe, it led to permanent discontinuation as often as bleeding at other sites overall, and more often than bleeding of comparable (minor) severity; this relationship is illustrated in Figure 3.

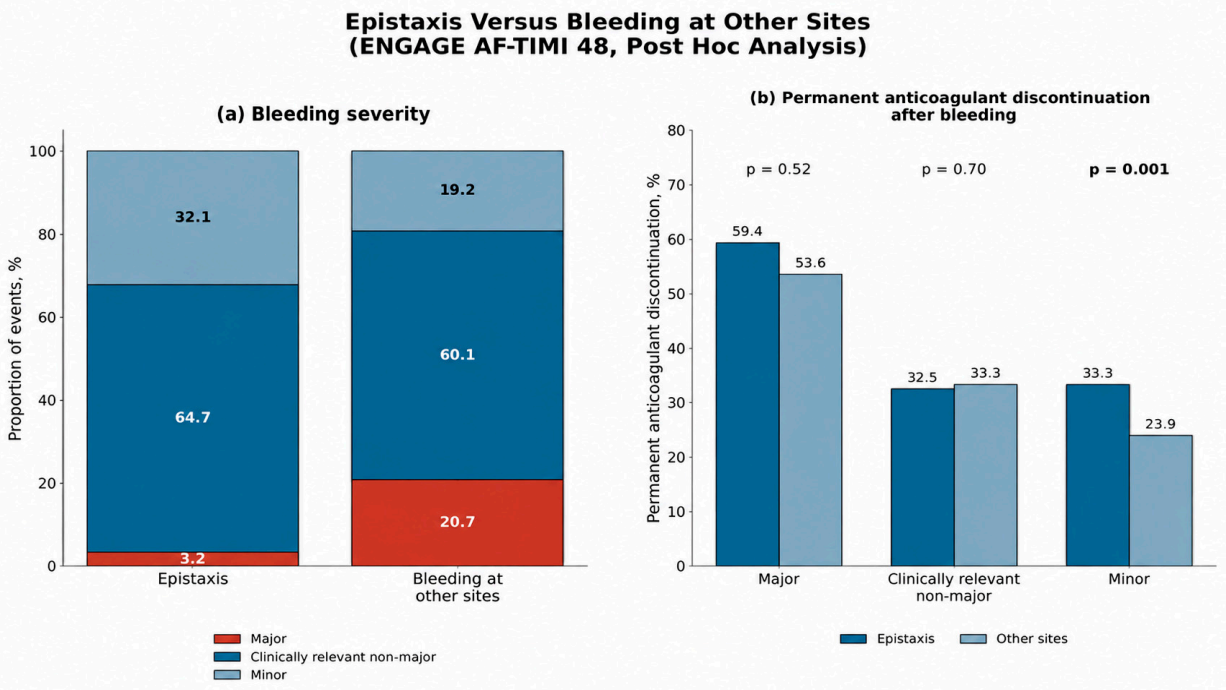


Figure 3. Severity of epistaxis and subsequent permanent anticoagulant discontinuation compared with bleeding at other sites (ENGAGE AF-TIMI 48).

Evidence regarding the need for intervention is less consistent. In a retrospective analysis of 470 cases, antithrombotic therapy was associated with an increased risk of severe epistaxis and hospitalization, whereas recurrence rates were unchanged [25]. In a cohort of 786 patients, age was the only independent predictor of the need for a minor intervention after multivariable adjustment [26]. In a series of 600 presentations, oral anticoagulation was associated with recurrence rather than with a severe course [8]. The impact of anticoagulant therapy on healthcare utilization has been examined in large administrative databases [27], and long-term outcomes have been described previously [28].

4. Pharmacological Characteristics of Antithrombotic Agents

Management of epistaxis depends on the drug class, the duration of its antithrombotic effect, and the availability of a specific reversal agent [29,30]. The principal characteristics are summarized in Table 3.

Table 3. Comparative pharmacological characteristics of antithrombotic agents.

Drug	Class and Mechanism	Plasma T½	Duration of Effect	Management of Epistaxis	Reversal
Acetylsalicylic acid, 75–100 mg once daily	Antiplatelet agent; irreversible COX-1 inhibition	15–20 min *	7–10 days, until renewal of the platelet pool *	Continue in minor and most CRNM bleeding; local hemostasis	No specific antidote. Platelet transfusion is not routinely indicated and should be considered only in selected life-threatening bleeding after multidisciplinary assessment [31,32]
Clopidogrel, 75 mg once daily	Antiplatelet prodrug; irreversible P2Y12 inhibition	6 h *	5–7 days *	When used as part of DAPT, modify or interrupt only jointly with the treating cardiologist	No specific antidote; platelet transfusion as for aspirin
Ticagrelor, 90 mg twice daily	Antiplatelet agent; reversible P2Y12 inhibition	7–9 h	3–5 days	Same approach; in selected patients the cardiologist may consider ticagrelor monotherapy after at least 1 month or de-escalation to clopidogrel; these strategies are not interchangeable	No approved specific reversal agent; platelet transfusion is of limited value while the drug is circulating
Warfarin	Vitamin K antagonist	20–60 h	Up to 5 days	Monitor INR; if above target without life-threatening bleeding, omit a dose; low-dose oral vitamin K only if bleeding requires intervention	Vitamin K (phytomenadione) and prothrombin complex concentrate
Dabigatran, twice daily	DOAC; direct thrombin inhibitor	12–17 h; prolonged when CrCl < 50 mL/min	12–24 h; longer in renal impairment	If not controlled locally, consider temporary withholding until hemostasis is secure; individualize duration by time since last dose and CrCl; INR not informative	Idarucizumab; prothrombin complex concentrate if unavailable
Rivaroxaban, once daily	DOAC; factor Xa inhibitor	5–9 h in younger patients; 11–13 h in older patients	12–24 h	If not controlled locally, consider temporary withholding until hemostasis is secure; INR not informative	Prothrombin complex concentrate; andexanet alfa only where still authorized (no longer marketed in the USA; thrombotic safety concerns, see text)
Apixaban, twice daily	DOAC; factor Xa inhibitor	~12 h	12–24 h	If not controlled locally, consider temporary withholding until hemostasis is secure; INR not informative	Prothrombin complex concentrate; andexanet alfa only where still authorized (no longer marketed in the USA; thrombotic safety concerns, see text)
Edoxaban, once daily	DOAC; factor Xa inhibitor	10–14 h	12–24 h	If not controlled locally, consider temporary withholding until hemostasis is secure; INR not informative	Prothrombin complex concentrate; andexanet alfa is not labeled for edoxaban (off-label use reported)

* For irreversible platelet inhibitors (acetylsalicylic acid and clopidogrel), the plasma elimination half-life should not be interpreted as the duration of the antiplatelet effect; platelet inhibition persists until the affected platelets are replaced. COX-1, cyclooxygenase-1; CrCl, creatinine clearance; CRNM, clinically relevant non-major; DAPT, dual antiplatelet therapy; DOAC, direct oral anticoagulant; INR, international normalized ratio; T½, plasma elimination half-life.

Management is determined not only by drug class but also by the clinical context, including the time elapsed since coronary stenting [4,5], the international normalized ratio (INR) [21], and renal function [33]. Reversal agents for dabigatran and factor Xa inhibitors have demonstrated efficacy in clinical studies [34,35]. In patients with stable coronary artery disease, treatment selection is addressed in a separate guideline [36].

The safety of andexanet alfa has, however, been questioned. In the randomized ANNEXA-I trial in factor Xa inhibitor-associated intracerebral hemorrhage, andexanet improved hemostatic efficacy compared with usual care (67.0% vs. 53.1%) but was associated with more thrombotic events (10.3% vs. 5.6%), including ischemic stroke (6.5% vs. 1.5%) [37]. After review of postmarketing data, the U.S. Food and Drug Administration concluded that the risks outweighed the benefits, and the manufacturer ended U.S. commercial sales on 22 December 2025; the product remains authorized in Europe and several other regions, so its availability is jurisdiction-specific [38,39]. Whether the thrombotic signal reflects the agent itself, the population studied, or the comparator, which consisted mostly of prothrombin complex concentrate, remains uncertain. At present, andexanet alfa can be considered for life-threatening bleeding associated with apixaban or rivaroxaban

only where it remains authorized and in accordance with its local label and regulatory status, with explicit consideration of thrombotic risk; it is not labeled for edoxaban, although off-label use has been reported. For epistaxis, which only rarely reaches this level of severity, these considerations apply to exceptional cases.

The availability of reversal agents also varies across healthcare systems and, as in other countries, depends on national registration: idarucizumab, a specific reversal agent for dabigatran [40], is widely registered, whereas in countries where andexanet alfa is not authorized, prothrombin complex concentrate is the only reversal option for factor Xa inhibitors.

5. Comparison of Current Clinical Guideline Recommendations

The management of epistaxis during antithrombotic therapy is addressed by documents that were developed independently and for different purposes. Table 4 compares five sets of guidance across 12 clinically relevant domains: AAO-HNS guidance [13], SFORL guidance [41], European Society of Cardiology guidelines [3,4], and ACC/AHA guidelines [5,42]. As in other countries, national clinical protocols are generally adapted from these international documents, and registry data show that their implementation in routine practice varies [43]; contemporary reviews of the management of acute coronary syndromes likewise draw on the same international recommendations [44]. No dedicated guideline on the management of epistaxis during antithrombotic therapy was identified.

Table 4. Comparison of current guideline recommendations for managing epistaxis during antithrombotic therapy.

Domain	AAO-HNS, 2020	SFORL, 2017	ESC, 2024 (AF) and 2023 (ACS)	ACC/AHA/ACCP/HRS, 2023 and ACC/AHA, 2025
Discontinuation of therapy in non-life-threatening bleeding	Local measures are first-line, before transfusion, reversal, or drug discontinuation	Before changing therapy, exclude overdose and assess thrombotic risk	Bleeding risk factors rarely justify discontinuation of anticoagulation	No epistaxis-specific recommendation
Dual antiplatelet therapy after stenting	Not addressed	Continue for 1 month after stenting and, if possible, for up to 3 months	Defined by ACS guidelines	At least 12 months as the default strategy in patients without high bleeding risk
Warfarin management with supratherapeutic INR	Not addressed	Corrective measures are determined by the INR; if bleeding remains uncontrolled, interrupt therapy, administer antidotes, and monitor INR	General recommendations	No INR thresholds specific to epistaxis
Reversal agents for DOACs	Reversal is mentioned without specifying individual agents	Published before reversal agents were incorporated into guidelines	Idarucizumab and andexanet alfa (Class IIa, Level B)	Idarucizumab and andexanet alfa are included; U.S. sales of andexanet alfa ended in December 2025 [38,39]
Bleeding-risk assessment	Not addressed	Assessment of thrombotic risk	Score-based assessment replaced by individualized evaluation of risk factors (Class I, Level B)	HAS-BLED retained
Choice of packing material	Absorbable materials in patients receiving antithrombotic therapy	Not detailed	Not addressed	Not addressed
Topical tranexamic acid	Not included in the key recommendations	Not addressed	Not addressed	Not addressed
Escalation: ligation and embolization	Included among interventions requiring outcome documentation	Modification of anticoagulant therapy is not recommended for embolization	Not addressed	Not addressed
Resumption of therapy	Not addressed	Not detailed	No timing recommendations specific to epistaxis	After major gastrointestinal bleeding, after correction of reversible causes (Class 2b); after intracranial hemorrhage in very-high-risk patients, after 1–2 weeks (Class 2a)

Table 4. *Cont.*

Domain	AAO-HNS, 2020	SFORL, 2017	ESC, 2024 (AF) and 2023 (ACS)	ACC/AHA/ACCP/HRS, 2023 and ACC/AHA, 2025
Combination of antiplatelet and anticoagulant therapy	Not addressed	Not detailed	Not recommended for stroke prevention (Class III, Level B)	Restricted to the period after acute coronary syndrome
Arterial hypertension	Limited guidance	Not addressed	Modifiable bleeding-risk factor	Blood pressure control is a component of management
Composition of the multidisciplinary team	Use of antithrombotic drugs should be documented	Joint decision-making	Interdisciplinary approach for non-modifiable risk factors	The decision to resume therapy is made by the team

6. Proposed Management Algorithm

Based on the evidence presented above and current consensus documents [13,20,21,30,41], we propose a stepwise algorithm for the management of patients with epistaxis during antithrombotic therapy (Figure 4). Because a single box cannot capture the heterogeneity of antithrombotic regimens, the antithrombotic step is expanded in two companion tables: a pragmatic stratification of thrombotic risk (Table 5) and agent-specific actions by severity (Table 6). The strata in Table 5 are a synthesis of guideline concepts for the purpose of this review and are not a validated score. The proposed algorithm is intended as a pragmatic framework for clinical decision-making rather than a validated management protocol. Recommendations supported by established guidelines are distinguished from extrapolations based on evidence from bleeding at other anatomical sites and from the authors' proposed operational criteria (see the evidence-basis note under Table 6). The thrombotic-risk strata and severity thresholds have not been prospectively validated in patients with epistaxis and should therefore support, rather than replace, individualized clinical assessment and multidisciplinary decision-making.

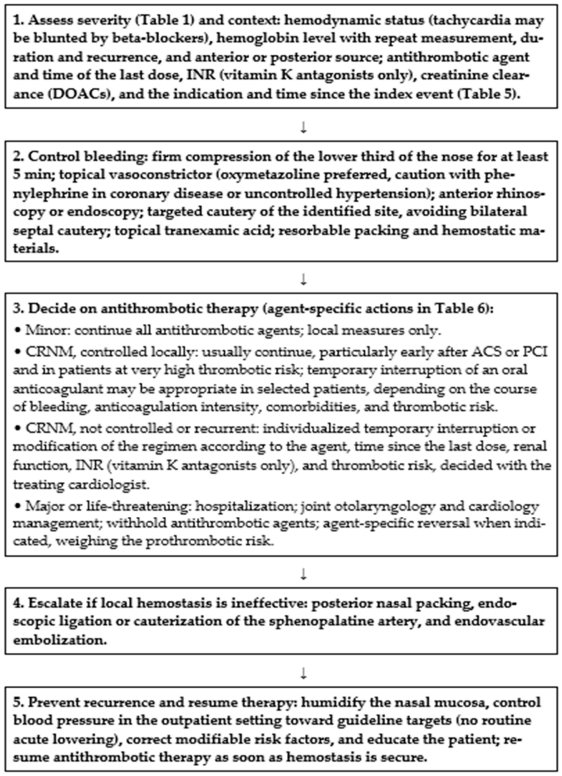


Figure 4. Algorithm for managing epistaxis during antithrombotic therapy. Severity tiers are defined in Table 1, thrombotic risk strata in Table 5, and agent-specific actions in Table 6. ACS, acute coronary syndrome; CRNM, clinically relevant non-major; DOAC, direct oral anticoagulant; INR, international normalized ratio; PCI, percutaneous coronary intervention.

Table 5. Pragmatic stratification of thrombotic risk for decisions on antithrombotic therapy during epistaxis.

Stratum	Examples by Indication	Implication for Management of Epistaxis
Very high	ACS or PCI within the past month [4,5]; ischemic stroke or transient ischemic attack within the past 3 months; venous thromboembolism within the past 3 months [20]; mechanical mitral valve, or mechanical aortic valve with additional thromboembolic risk factors [45]; triple-positive antiphospholipid syndrome treated with a vitamin K antagonist [46]	Avoid interruption whenever possible; prefer early escalation of otolaryngologic hemostasis (posterior packing, sphenopalatine artery ligation, embolization) over pharmacological reversal; if interruption is unavoidable, keep it as short as possible and decide jointly with the cardiologist or neurologist
High	ACS or PCI 1–12 months earlier [4,5]; atrial fibrillation with prior stroke or transient ischemic attack more than 3 months earlier or with several additional risk factors [3]; venous thromboembolism 3–12 months earlier	Continue if bleeding is controlled locally; for uncontrolled CRNM bleeding, prefer modification (for example, withdrawal of aspirin from combination therapy, dose review) over complete interruption; if interrupted, resume as soon as hemostasis is secure
Low to moderate	Remote ACS or PCI (more than 12 months) on single antiplatelet therapy; secondary prevention after a remote stroke; atrial fibrillation with few risk factors; extended anticoagulation after provoked venous thromboembolism	Temporary interruption until hemostasis is secure is acceptable for uncontrolled CRNM or major bleeding; the episode is an opportunity to review the indication and dose [20]

This stratification is a synthesis for the purpose of this review and not a validated score. For example, a patient taking clopidogrel for secondary prevention after a stroke 3 years earlier falls into the low-to-moderate stratum, whereas the same patient 1 week after stroke falls into the very-high stratum; a patient taking warfarin for a mechanical mitral valve or triple-positive antiphospholipid syndrome is at a much higher thrombotic risk than a patient taking warfarin for atrial fibrillation with a CHA_2DS_2-VASc score of 2. ACS, acute coronary syndrome; CRNM, clinically relevant non-major; PCI, percutaneous coronary intervention.

Table 6. Agent-specific management of antithrombotic therapy according to epistaxis severity.

Agent (Dosing)	Minor	Clinically Relevant Non-Major	Major or Life-Threatening
Acetylsalicylic acid (once daily)	Continue	Continue with local hemostasis. If bleeding is not controlled and the patient receives combination therapy, the cardiologist may consider withdrawing aspirin in selected patients, according to ischemic and bleeding risk rather than automatically [3–5]	Withhold. No specific antidote; platelet transfusion is not routine and is considered only in selected life-threatening bleeding after multidisciplinary assessment [31,32]
P2Y12 inhibitor alone or in DAPT (clopidogrel or prasugrel once daily; ticagrelor twice daily)	Continue	Continue when bleeding is controlled locally, especially within 1 month of ACS or PCI. If bleeding recurs despite otolaryngologic management, the cardiologist may consider modifying rather than stopping therapy. In selected patients who have tolerated DAPT after ACS and PCI, guideline-supported options include transition to ticagrelor monotherapy after at least 1 month or de-escalation to clopidogrel; these strategies are not interchangeable and are chosen according to ischemic and bleeding risk, the P2Y12 inhibitor used, and PCI complexity, not applied automatically because of epistaxis [4,5]	Withhold the agent or agents chosen jointly with the cardiologist, weighing the risk of stent thrombosis; no approved reversal agent; resume as soon as hemostasis is secure
Warfarin or other vitamin K antagonist	Continue; check INR if not recently measured	Check INR. INR in range: continue. INR above range: omit one or more doses until the INR returns to range; low-dose oral vitamin K may be used when bleeding requires intervention, avoiding overcorrection [20,47]	Withhold; intravenous vitamin K and four-factor prothrombin complex concentrate [20]

Table 6. *Cont.*

Agent (Dosing)	Minor	Clinically Relevant Non-Major	Major or Life-Threatening
Dabigatran (twice daily; predominantly renal elimination)	Continue	If bleeding is not controlled locally or recurs, consider temporary withholding until hemostasis is secure; individualize the duration according to the time since the last dose, renal function (the effect is prolonged when creatinine clearance is below 50 mL/min), interacting drugs, and thrombotic risk; INR is not informative [20,30]	Withhold; idarucizumab; prothrombin complex concentrate if idarucizumab is unavailable [20,30]
Factor Xa inhibitors (apixaban twice daily; rivaroxaban and edoxaban once daily)	Continue	If bleeding is not controlled locally or recurs, consider temporary withholding until hemostasis is secure; individualize the duration according to the time since the last dose, renal function, interacting drugs, the indication for anticoagulation, and whether bleeding continues; INR is not informative [20,30]	Withhold; four-factor prothrombin complex concentrate; andexanet alfa only where it remains authorized and according to its local label, given the excess of thrombotic events in ANNEXA-I and the end of its U.S. commercial sales in December 2025 [20,37–39]; activated charcoal if the last dose was taken within a few hours [30]

Local hemostasis comes first in every tier (Figure 4). Thrombotic risk (Table 5) modifies every column: in the very-high stratum, interruption should be avoided or limited to the shortest possible period and decided jointly with the cardiologist; in the low-to-moderate stratum, temporary interruption until hemostasis is secure is acceptable. Therapy should be resumed as soon as hemostasis is secure, because prolonged interruption carries thrombotic risk [10,20]. Evidence basis: continuation of therapy in minor bleeding with local hemostasis first, management of vitamin K antagonists by INR and vitamin K, and DOAC withholding guided by the last dose and renal function are taken from guidelines and consensus documents on antithrombotic-associated bleeding in general [13,20,30,47]; aspirin withdrawal and P2Y12 inhibitor de-escalation are guideline-supported for ACS and PCI [4,5] and are extrapolated to epistaxis; recommendations on reversal agents are based on guidelines and on trials in bleeding at other sites [20,30,34,35,37]; and the assignment of these actions to the severity tiers of Table 1 and the thrombotic risk strata of Table 5 is a pragmatic proposal of the authors. ACS, acute coronary syndrome; DAPT, dual antiplatelet therapy; INR, international normalized ratio; PCI, percutaneous coronary intervention.

In practical terms, minor epistaxis and CRNM epistaxis controlled by first-line local measures usually do not require any change in antithrombotic therapy. Routine reversal is not appropriate for non-major bleeding; however, temporary interruption of an oral anticoagulant may be appropriate in selected patients until clinical stability and hemostasis are achieved, depending on the severity and course of bleeding, the intensity of anticoagulation, comorbidities, and thrombotic risk [20]. Interruption or modification should be considered when bleeding persists or recurs despite compression, a vasoconstrictor, targeted cautery, and anterior packing; when posterior packing or surgical or endovascular hemostasis is required; when hemoglobin decreases by 20 g/L or more or transfusion is needed; when there is hemodynamic compromise; when the INR is above the therapeutic range; or when there is concomitant major bleeding at another site. Even then, the extent and duration of interruption depend on the thrombotic risk stratum (Table 5).

Local hemostatic measures remain the first-line approach to bleeding control regardless of whether antithrombotic therapy is continued [13]. Topical tranexamic acid has been studied most extensively: in the randomized NoPAC trial, it did not reduce the need for nasal packing [48], whereas meta-analyses and a network meta-analysis demonstrated efficacy compared with other noninvasive approaches [49,50]; a separate randomized trial specifically enrolled patients receiving antiplatelet therapy [51].

These findings are less contradictory than they appear. NoPAC-enrolled patients whose bleeding persisted despite at least 10 min of first aid and a topical vasoconstrictor, applied 200 mg of tranexamic acid on a dental roll against a placebo given against the same background, and used anterior packing at any time during the visit as a pragmatic primary outcome (43.7% vs. 41.3%) [48]. Most trials pooled in the meta-analyses used higher doses, typically 500 mg on cotton pledgets or packing, compared tranexamic acid with packing or with a vasoconstrictor alone, and measured early cessation of bleeding [49,50]. In the

trial by Zahed et al. in patients taking antiplatelet agents, 500 mg on a pledget stopped bleeding within 10 min in 73% of patients versus 29% with conventional anterior packing and allowed discharge within 2 h in 97% versus 13% [51]. Differences in dose, mode of application, background vasoconstrictor use, and outcome definitions are therefore the most plausible explanation. Topical tranexamic acid can reasonably be used as an adjunct in patients receiving antithrombotic therapy, while acknowledging that it has not been shown to reduce the need for packing.

Two widely used local measures deserve specific comment in this population. Topical vasoconstrictors facilitate inspection and cautery, and oxymetazoline achieved control in about two-thirds of patients in an early series [52], but these agents are absorbed through the nasal mucosa. Phenylephrine in particular has caused severe hypertension, reflex bradycardia, and pulmonary edema when applied in large amounts, which led to dose-limiting recommendations [53]. In patients with coronary artery disease, recent acute coronary syndrome, or uncontrolled hypertension, we therefore suggest oxymetazoline in the smallest effective amount on a pledget, avoidance of repeated large-volume application, and monitoring of blood pressure and heart rate. Anticoagulation does not contraindicate cautery, but chemical cautery with silver nitrate is effective only on a field that is not actively bleeding, so temporary control with compression or a vasoconstrictor is needed first. Cautery should be restricted to the identified bleeding point [13], and bilateral septal cautery in one session should be avoided because of the risk of septal perforation. Because oral anticoagulation is associated with recurrence rather than with a severe course [8], repeat cautery is needed more often in this population, and endoscopic localization is recommended for recurrent bleeding [13]. When packing is required, resorbable materials are preferred in patients taking anticoagulant or antiplatelet agents, because removal of nonresorbable packs traumatizes the mucosa and provokes rebleeding [13]. The choice of packing material has also been evaluated in a meta-analysis [54]. Refractory bleeding requires stepwise escalation [55,56], including endovascular embolization [57,58].

7. Clinical Cases

The following two cases are real, retrospectively selected clinical cases obtained from the medical records of patients who received routine clinical care at the Department of Otolaryngology, JSC Astana Medical University, Astana, Kazakhstan (Case 1), and the City Cardiology Center, Almaty, Kazakhstan (Case 2). All patient information was fully de-identified, and any details that could permit patient identification were removed. The cases are presented solely for illustrative and educational purposes to demonstrate the practical application of the management principles discussed in this narrative review. They are not intended to provide evidence of treatment effectiveness or to constitute a separate clinical study.

7.1. Epistaxis During Warfarin Therapy

A 72-year-old man. Diagnoses: persistent atrial fibrillation; coronary artery disease; grade 2 arterial hypertension. Treatment: warfarin 5 mg/day (target INR, 2.0–3.0), bisoprolol, and perindopril. He presented to the otolaryngology department with recurrent right-sided epistaxis over 3 days. On examination, blood pressure was 160/90 mmHg, and the bleeding source was localized to Kiesselbach's plexus on the right. Laboratory findings showed hemoglobin 118 g/L and INR 4.8.

Management: anterior nasal packing; warfarin withheld for 48 h; phytomenadione (vitamin K1) 1 mg orally as a single dose. After 24 h, the INR decreased to 2.9 and bleeding ceased. Warfarin was restarted at a reduced dose of 4.375 mg/day, and the INR 24 h later was 2.8.

Comment: The INR of 4.8 was above the therapeutic range and was accompanied by recurrent CRNM epistaxis that required anterior packing. For an INR in this range without clinically significant bleeding, omitting or reducing warfarin doses is usually sufficient, and routine vitamin K is not recommended [47]. Low-dose oral vitamin K was therefore an individualized decision of the treating team rather than a standard response to an INR of 4.8. It was chosen because bleeding had recurred over 3 days and required intervention, so that a faster but controlled return to the therapeutic range was desired to reduce the risk of further packing. This is consistent with the 2020 ACC expert consensus, which distinguishes bleeding that requires intervention from an elevated INR without clinically significant bleeding and supports vitamin K for bleeding associated with a vitamin K antagonist that requires intervention [20]. A low oral dose was chosen to avoid overcorrection and to allow early resumption of anticoagulation in a patient with atrial fibrillation, and the INR returned to the therapeutic range within 24 h without falling below it. Blood pressure control was an important component of management because arterial hypertension is a modifiable bleeding-risk factor [59].

7.2. Recurrent Epistaxis During Dual Antiplatelet Therapy

A 58-year-old woman. Diagnoses: coronary artery disease; non-ST-segment elevation acute coronary syndrome. Stenting of the left anterior descending coronary artery had been performed 14 days earlier. Treatment: acetylsalicylic acid 100 mg and ticagrelor 90 mg twice daily. She presented with daily low-volume epistaxis for 1 week.

Management: Cardiology assessment concluded that early discontinuation of antiplatelet therapy after stenting was not indicated; otolaryngology management consisted of cauterization of vessels in Kiesselbach's plexus with 10% silver nitrate solution and intranasal dexpanthenol ointment twice daily. Outcome: Bleeding ceased within 2 weeks, and antiplatelet therapy was continued without modification.

Comment: In the early period after acute coronary syndrome, prevention of stent thrombosis takes priority [4,5]. The case also shows where this approach ends: the bleeding was CRNM and could be controlled locally. Had it recurred despite cautery and resorbable packing, the next step would have been an individualized decision with the treating cardiologist to modify rather than stop antiplatelet therapy, for example, after the first month and in the absence of high ischemic risk, by transition to ticagrelor monotherapy or by de-escalation to clopidogrel, with the strategy chosen according to her ischemic and bleeding risk rather than applied automatically (Table 6) [4,5].

8. Discussion

First, continuation of antithrombotic therapy should be prioritized whenever bleeding can be controlled locally. The harm of unnecessary interruption is the main argument for this position. The best quantitative estimate comes from the GARFIELD-AF registry, which was not specific to epistaxis: interruption of oral anticoagulation for 7 or more consecutive days was associated with a higher risk of ischemic stroke and systemic embolism (hazard ratio, 2.21; 95% CI, 1.42–3.44) and of all-cause death (1.62; 1.25–2.09), even minor bleeding nearly doubled the likelihood of interruption, and most patients who interrupted therapy were still untreated 30 days later [10]. Because ENGAGE AF-TIMI 48 shows that minor epistaxis in particular leads to permanent discontinuation [2], these registry data indicate what is at stake when epistaxis prompts interruption, although they cannot be attributed to epistaxis directly.

For most minor or clinically manageable episodes of epistaxis, local control of bleeding should therefore precede any consideration of antithrombotic interruption, particularly in patients with atrial fibrillation and a high risk of thromboembolic complications [60]

and during the early period after coronary stenting [4,5]. This principle is not universal. Modification or temporary interruption of antithrombotic therapy is appropriate in hemodynamic instability, major bleeding, uncontrolled posterior epistaxis, a clinically important decrease in hemoglobin, supratherapeutic anticoagulation, concomitant intracranial or other major bleeding, and failure of local measures, and it should be individualized according to thrombotic risk [20,21,42].

Second, management should be individualized according to the drug. For warfarin, the INR guides management. For direct oral anticoagulants, the INR is unreliable, and decisions depend on the time since the last dose, renal function, the specific agent and its dosing schedule, the dose, interacting drugs, and the likelihood of continued absorption [30]. A fixed instruction to omit one or two doses is therefore ambiguous: one omitted dose interrupts anticoagulation for about 24 h with once-daily rivaroxaban or edoxaban but for only about 12 h with twice-daily apixaban or dabigatran, and the effect of dabigatran persists considerably longer when creatinine clearance is below 50 mL/min. The same consideration applies to antiplatelet agents taken twice daily, such as ticagrelor, or once daily, such as clopidogrel. For CRNM bleeding, temporary interruption of a DOAC may be considered according to bleeding severity, time since the last dose, renal function, and thrombotic risk, whereas routine interruption is unnecessary for minor epistaxis controlled by local measures. In life-threatening or uncontrolled bleeding, specific reversal or nonspecific prohemostatic therapy should be considered according to the anticoagulant involved, time since the last dose, renal function, availability of reversal agents, and thrombotic risk [20,30]. Direct oral anticoagulants are associated with a more favorable epistaxis profile than warfarin [1,22], although differences between treatment regimens did not remain statistically significant after multivariable adjustment in some cohorts [26].

Third, antiplatelet therapy after coronary events requires particular nuance, because the period after ACS or PCI is not a single clinical situation. The 2025 ACC/AHA guideline retains 12 months of DAPT as the default after ACS in patients without high bleeding risk, but it also recognizes evidence-based alternatives, including transition to ticagrelor monotherapy after at least 1 month and shorter or de-escalated regimens in patients at high bleeding risk [5]. These are distinct strategies supported in selected patients who have tolerated DAPT, not interchangeable responses to any bleeding episode; recurrent epistaxis is a reason to reassess the regimen with the cardiologist rather than to apply one of them automatically [5,44]. The 2023 ESC guideline on ACS and the 2024 ESC guideline on chronic coronary syndromes similarly support individualized abbreviation or de-escalation of DAPT on the basis of ischemic and bleeding risk [4,36]. For clinically significant epistaxis during DAPT, continuation of antiplatelet therapy should generally be considered when bleeding can be controlled locally, particularly during the early period after ACS or PCI. The decision to interrupt, de-escalate, or otherwise modify DAPT should be individualized according to bleeding severity and persistence, ACS versus chronic coronary syndrome, time since PCI, stent characteristics and procedural complexity, ischemic and stent-thrombosis risk, and the indication for antiplatelet therapy, preferably in consultation with the treating cardiologist.

Fourth, multidisciplinary collaboration is essential. In GARFIELD-AF, the decision to discontinue therapy was made by the treating physician in 51.0% of cases and by the patient alone in only 18.5% [10]. The ENGAGE AF-TIMI 48 analysis demonstrates the same pattern specifically for epistaxis [2]. This leads to a practical conclusion: effective local hemostasis performed by an otolaryngologist can reduce the need to interrupt antithrombotic therapy and may thereby plausibly reduce exposure to thrombotic risk. This remains an inference: the GARFIELD-AF data concern anticoagulant interruption in a

broader population with atrial fibrillation, and no study has shown that an otolaryngologic intervention independently reduces stroke, myocardial infarction, or stent thrombosis.

Fifth, comparison of the guidance documents (Table 4) reveals three clinically important areas of discordance. The period of protected DAPT ranges from 1 month in the SFORL document [41] to a 12-month default in the ACC/AHA guideline, which nevertheless allows earlier modification in selected patients [5], whereas the ESC guidelines support individualized abbreviation or de-escalation of DAPT [4]. Approaches to bleeding-risk assessment also differ: individualized assessment of risk factors [3] and the HAS-BLED score [42,61] are used concurrently. Finally, otolaryngology and cardiology documents frame the sequencing of local hemostasis and antithrombotic interruption in opposite ways.

Sixth, as in other countries, national clinical protocols are usually adapted from cardiology guidelines, and such adaptations may not address bleeding of otolaryngologic origin. Where protocols are developed without otolaryngology input, the multidisciplinary team for hemorrhagic complications may not include an otolaryngologist, source control may be described only in terms of gastrointestinal endoscopy, and reversal agents may be recommended without regard to their national availability [40]. Registry data on antithrombotic therapy from different countries [43,62] make it feasible to assess such gaps and the real-world use of antithrombotic agents.

Prevention of recurrence includes nasal mucosal humidification, treatment of arterial hypertension, patient education, and, in selected patients and at the cardiologist's discretion, de-escalation of antiplatelet therapy [4,5,59]. In patients with recurrent major bleeding who have an absolute indication for anticoagulation, left atrial appendage closure may be considered [3,63]. Conditions and factors associated with epistaxis in patients receiving antithrombotic therapy, and their implications for management, are summarized in Table 7.

Table 7. Conditions and factors associated with epistaxis in patients receiving antithrombotic therapy.

Category	Condition or Factor	Relevance for Patients Receiving Antithrombotic Therapy
Antithrombotic therapy	Vitamin K antagonists, particularly with a supratherapeutic INR; direct oral anticoagulants; antiplatelet agents and their combinations	Warfarin is associated with higher rates of clinically relevant epistaxis than DOACs; combination antiplatelet therapy increases the need for intervention [1,7,22,26]
Drug exposure	Reduced creatinine clearance; interacting drugs	Prolong the anticoagulant effect of DOACs, particularly dabigatran, and the period of withholding needed [30,33]
Cardiovascular comorbidity	Arterial hypertension; atherosclerotic cardiovascular disease	Hypertension is associated with a higher incidence and severity of epistaxis [64,65]; epistaxis is also associated with atherosclerotic disease [6]
General bleeding-risk factors	Uncontrolled hypertension, labile INR, renal or hepatic dysfunction, prior bleeding, alcohol excess, older age	Components of HAS-BLED and of individualized bleeding-risk assessment; modifiable factors should be corrected [3,59,61]; age predicts the need for intervention [26]
Local nasal factors	Dry nasal mucosa, digital trauma, septal deformity, and other local mucosal factors	Common precipitants of anterior epistaxis; addressed by humidification, local treatment, and patient education [13]
Vascular malformations	Hereditary hemorrhagic telangiectasia	Causes recurrent epistaxis and complicates anticoagulation; left atrial appendage closure may be considered in atrial fibrillation [63]; outside the scope of this review

DOAC, direct oral anticoagulant; INR, international normalized ratio.

Arterial hypertension deserves a more precise statement. A meta-analysis of 10 studies found an association between hypertension and epistaxis (odds ratio, 1.53; 95% CI, 1.18–1.99) [64], and in a nationwide Korean cohort, hypertension was associated with a higher incidence of epistaxis (adjusted hazard ratio, 1.47; 95% CI, 1.30–1.66) and with more severe presentations requiring emergency care or posterior packing, but not with a statistically significant increase in recurrence (incidence rate ratio, 1.23; 95% CI, 0.77–2.00) [65]. Elevated blood pressure at presentation is also partly a stress response to the bleeding. We found no study showing that blood pressure control specifically reduces recurrence of epistaxis, so the evidence for this effect is indirect. Acute blood pressure lowering is not recommended as routine treatment of active epistaxis, because its benefit is unproven

and excessive reduction may cause cerebral, coronary, or renal ischemia [13]. Sustained blood pressure control is nevertheless justified on general grounds: uncontrolled systolic blood pressure is a modifiable bleeding-risk factor in patients receiving anticoagulants [59], and current ESC guidance recommends a systolic target of 120–129 mmHg in most treated adults, provided that it is tolerated [66]. In patients with recurrent epistaxis and hypertension, we therefore recommend reassessing blood pressure after the bleeding has settled, confirming hypertension with out-of-office measurement, and intensifying antihypertensive therapy toward the guideline target, rather than lowering blood pressure acutely during the bleeding episode.

A limitation inherent in the bleeding scales themselves deserves mention. In the Bleeding Academic Research Consortium definitions, type 3b includes bleeding requiring surgical intervention for control but explicitly excludes dental, nasal, skin, and hemorrhoidal bleeding [18]. Epistaxis that requires arterial ligation or embolization therefore does not qualify as type 3b and is classified as less severe unless the hemoglobin, transfusion, or vasoactive-support thresholds are reached. This does not mean that BARC necessarily underestimates the severity of every episode requiring arterial ligation or embolization; rather, it illustrates a limitation in mapping epistaxis to standard bleeding classifications, because BARC categories, hemoglobin decline, transfusion, hemodynamic compromise, and the need for a procedure capture different dimensions of bleeding severity, and it limits the comparability of published rates. For this reason, Table 1 treats the need for surgical or endovascular hemostasis as a major criterion within a pragmatic, epistaxis-specific framework whose categories should not be regarded as validated equivalents of BARC or ISTH major bleeding. Hemodynamic criteria have a similar limitation: systolic blood pressure may remain normal until a substantial part of the circulating volume has been lost, and in an analysis of 36,504 trauma patients, only 9.3% could be assigned to an ATLS shock class when heart rate, systolic blood pressure, and the Glasgow Coma Scale were considered together [67]. In patients taking beta-blockers, which are common in this population, tachycardia as an early sign of hypovolemia is further blunted, which is why repeated hemoglobin measurement is included in the initial assessment (Figure 4).

Limitations

Large randomized trials specifically addressing the management of epistaxis during antithrombotic therapy are lacking; most recommendations are based on expert opinion and retrospective data [29]. The present work is a narrative review and therefore cannot exclude selection bias in source identification. The guideline comparison reflects versions in force at the time of the last search (end of August 2026). The severity classification in Table 1 and the thrombotic risk strata in Table 5 are pragmatic syntheses of existing definitions and guideline concepts. They have not been prospectively validated for epistaxis, and their usefulness should be tested in registries that record both bleeding severity and decisions on antithrombotic therapy. Most agent-specific actions in Table 6 are extrapolated from guidance on antithrombotic-associated bleeding in general or from trials of bleeding at other sites rather than derived from studies of epistaxis (see the evidence-basis note under Table 6). The clinical cases are illustrative and do not provide evidence of effectiveness.

9. Conclusions

Epistaxis is a common and clinically important problem in patients with cardiovascular disease: it is generally less severe than bleeding at other sites but disproportionately often leads to treatment discontinuation. For minor and locally controlled CRNM epistaxis, routine interruption of antithrombotic therapy is not indicated; interruption, de-escalation, or reversal should be reserved for uncontrolled or major bleeding and individualized ac-

cording to the agent, renal function, the time since the last dose, and thrombotic risk. Local hemostatic measures are central to management, because effective local hemostasis can help avoid unnecessary interruption of antithrombotic therapy, although its effect on thrombotic outcomes has not been directly demonstrated. Consistent use of operational severity criteria would make both clinical decisions and published data comparable. Bleeding-risk assessment should focus on identifying modifiable factors, including sustained blood pressure control. As in other countries, national adaptations of international guidelines should include an otolaryngologist in the multidisciplinary team and explicitly address hemostatic methods for bleeding of otolaryngologic origin.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/diseases14110414/s1, Figure S1: Central Illustration. Integrated clinical approach to epistaxis during antithrombotic therapy; Table S1: Principal conditions and factors associated with epistaxis.

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Abbreviations

AAO-HNS	American Academy of Otolaryngology-Head and Neck Surgery
ACC/AHA	American College of Cardiology/American Heart Association
ACC/AHA/ ACCP/HRS	American College of Cardiology/American Heart Association/American College of Chest Physicians/Heart Rhythm Society
ACC/AHA/ACEP/ NAEMSP/SCAI	American College of Cardiology/American Heart Association/American College of Emergency Physicians/National Association of EMS Physicians/Society for Cardiovascular Angiography & Interventions
ACS	acute coronary syndrome
AF	atrial fibrillation
ANNEXA-I	Andexanet Alfa, a Novel Antidote to the Anticoagulation Effects of Factor Xa Inhibitors, in Intracerebral Hemorrhage trial
ATLS	Advanced Trauma Life Support
BARC	Bleeding Academic Research Consortium
CHA_2DS_2-VASc	Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Stroke or transient ischemic attack, Vascular disease, Age 65–74 years, Sex category
CI	confidence interval
COX-1	cyclooxygenase-1
CrCl	creatinine clearance
CRNM	clinically relevant non-major
DAPT	dual antiplatelet therapy
DOAC	direct oral anticoagulant
ENGAGE AF-TIMI 48	Effective Anticoagulation with Factor Xa Next Generation in Atrial Fibrillation-Thrombolysis in Myocardial Infarction 48
ESC	European Society of Cardiology
GARFIELD-AF	Global Anticoagulant Registry in the FIELD-Atrial Fibrillation
GUSTO	Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries
HAS-BLED	Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly, Drugs/alcohol concomitantly
INR	international normalized ratio
ISTH	International Society on Thrombosis and Haemostasis
$MAQI^2$	Michigan Anticoagulation Quality Improvement Initiative
MEDLINE	Medical Literature Analysis and Retrieval System Online
NoPAC	The Use of Tranexamic Acid to Reduce the Need for Nasal Packing in Epistaxis trial
P2Y12	purinergic receptor P2Y12
PCI	percutaneous coronary intervention
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
SANRA	Scale for the Assessment of Narrative Review Articles
SFORL	French Society of Otorhinolaryngology
T½	plasma elimination half-life

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